

## BUILDING-RELATED ILLNESS IN OCCUPANTS OF MOLD-CONTAMINATED HOUSES: A CASE SERIES

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### ABSTRACT

The clinical presentation and course of individuals and families who developed illnesses related to non-infectious fungal exposures inside their homes is described. Occupants developed their illnesses shortly after their homes had been water damaged. A few occupants had a specific building-related illness, such as hypersensitivity pneumonitis or asthma exacerbation, but most had a "sick building syndrome" symptom complex involving irritation/inflammation of the mucous membranes, respiratory tract, and skin; fatigue; and/or neurocognitive dysfunction. All cases required months or years to correctly diagnose. Air, surface, and/or bulk microbiological sampling in most of the homes yielded high concentrations of toxigenic fungi, including *Stachybotrys chartarum* and *Penicillium* and *Aspergillus* species, emanating from water-damaged building materials. Most of the ill individuals had complete clinical improvement shortly after their removal from the contaminated indoor environment, but a few individuals continued to experience symptoms in response to a variety of environmental irritants. The author proposes a new clinical syndrome entity to describe the non-infectious, mold-related, building-related illness.

### KEY WORDS:

Building-related illness, sick building syndrome, toxigenic fungi, environmental disease, *Stachybotrys chartarum*, hypersensitivity pneumonitis.

### INTRODUCTION

Building-related illnesses (BRIs) have been recognized for many years as occupational or environmental diseases. Specific disease entities such as asthma, hypersensitivity pneumonitis (HP) (Banaszak et al. 1970; Bernstein et al. 1983), and legionellosis have been described in relation to a number of chemical, allergenic and infectious agents. The "sick building syndrome" (SBS), in contrast, remains a clinical and epidemiological entity for which a specific toxicological, microbiological, allergic or other etiology has not been determined clinically or environmentally (Hodgson 1995). In SBS, a significant percentage (undefined) of building occupants complains of a combination of respiratory, mucous membrane, constitutional, skin, and/or neurocognitive symptoms that occur predictably when the occupants are inside the building environment and which improve when they are away from the building. "Multifactorial" etiologies, including inadequate ventilation (Stenberg et al. 1994), off-gassed organic vapors from building materials, and psychogenic factors (Skov et al. 1989), have been implicated in published

cross-sectional and retrospective epidemiological studies of such "sick" buildings, but to date no unifying etiology has been demonstrated. Air quality investigations of buildings where occupants complain of these unexplained, building-related symptoms have typically focused on sampling for chemical (e.g., carbon monoxide, formaldehyde, fiberglass), allergic, and infectious agents (Menzies et al. 1996; Hodgson 1995). In most such cases, no abnormal contaminant is found and the building is labeled as "sick," suggesting an idiopathic etiology. Cases and studies of "sick" buildings published in the medical literature have mostly involved commercial office buildings and manufacturing facilities (Burge et al. 1987), with fewer reported cases of residential dwellings (Croft et al. 1986).

The role of water damage to and fungal colonization of building materials, with detection of fungi in air and/or on surfaces at high concentrations, is increasingly being recognized as a specific cause of some BRIs (notably asthma and HP), and possibly the sick building syndrome itself (Hodgson et al. 1998; Johanning et al. 1996; Sudakin 1998). In some "sick" buildings, external or internal water damage may not be recognized or detected until after an initial air quality investigation for non-microbiological causes has been completed. Several recently published clinical-epidemiological studies of buildings with occupants with SBS symptoms have identified the presence of high airborne or surface concentrations of fungal spores, trichothecene mycotoxins, and microbiological volatile organic chemicals (MVOC) produced by certain fungi which are associated with clinical symptomatology (Johanning et al. 1996). However, large gaps in clinical, pathological, epidemiological, and exposure assessment remain in determining the causal relationship between indoor fungal contamination and the SBS (Smoragiewicz et al. 1993).

## **MATERIALS AND METHODS**

Five (5) cases evaluated in private occupational/environmental medicine (OEM) consultation practice by the author are presented. The cases are comprised of individuals or families who were either self-referred (n=2), referred by an industrial hygienist (n=2), or referred for evaluation by a workers' compensation insurer (n=1) for consultation. The history of illness, its relationship to the home environment, the medical evaluation(s) and treatment(s), and past medical, occupational and environmental history were obtained for each case. The history of building-related water damage, if known, was reviewed. Walk-through inspection of the homes was performed by the author in 4 of 5 cases. Environmental microbiological testing of surface, bulk, and air samples, either before or after the clinical evaluation, was obtained in all cases. Occupants' clinical response to removal from the mold-contaminated home environment were documented.

## **RESULTS**

Results of the five cases are summarized in the Tables (Cases #1-5) below. All of the cases occurred in Nevada or Utah. Details of the clinical and microbiological investigations for each case are available upon request from the author.

**TABLE I:** Mold-Contaminated Houses - A Case Series.

<b>Case</b>	1
<b>Subject(s)</b>	78 year old, single woman
<b>Description of Home</b>	50 year old, 3-story house
<b>Structural Damage to Home</b>	Plumbing fixture burst on top floor, flooding walls, ceilings, carpets, floors. Two inches of standing water has been in basement. Partial remediation occurred without containment. Not all wet materials were removed; others were painted over or sprayed with fungicide.
<b>Clinical Illness</b>	Subject had a history of stable bronchiolitis obliterans with mild, chronic cough. After entering the partially-remediated house, she developed immediate symptoms of fatigue, weakness, sore throat, chest pressure, and nasal congestion, followed by delayed-onset, flu-like illness (fevers, chills, cough, dyspnea) that worsened after several hours in the house.. Re-entry challenge reproduced fever, chills, dyspnea and chest pressure symptoms, with new infiltrates on chest x-ray and hypoxemia.
<b>Duration of Symptoms before Diagnosis</b>	6 months
<b>Microbiological Sampling Results</b>	Surface wipe sampling of dust throughout the house demonstrated a variety of atypical culturable fungi of various species, notably <i>Penicillium auranteogriseum</i> , with maximum concentration of $1.7 \times 10^6$ CFU/in <sup>2</sup> (75% predominant) in basement.
<b>Diagnosis</b>	HP; "SBS-like illness"
<b>Intervention and Outcome</b>	Removed from home, upon which symptoms resolved. Only baseline cough persisted.
<b>Comments</b>	Homeowners' insurance company insisted home was habitable. Subject initiated a lawsuit against homeowners' insurance company and remediation contractor.
<b>Case</b>	2
<b>Subject(s)</b>	40 year old, male, employed as a heavy equipment mechanic, and his family (wife 38, two teenage children)
<b>Description of Home</b>	Rental, one-story townhouse, 10 years old. Family was not aware of any history of water damage to the house.
<b>Structural Damage to Home</b>	Subject and family noted only black discoloration underneath linoleum in the bathroom upon moving into townhouse; they did not realize there was water damage. Bathroom and adjacent walls were extensively water damaged. Subsequently extensive water damage to subfloor and standing water in crawl space was detected. The cause of water damage was chronic plumbing leaks and surface infiltration of underground aquifer.
<b>Clinical Illness</b>	Subject complained of persistent sinus congestion, rhinitis, sore throat, swollen cervical lymph nodes, cough productive of brown and green sputum, fevers with night sweats, and profound, unex-

Table I cont.

	<p>plained fatigue. Dyspnea, cough, and night sweats became predominant. He had one chest x-ray at the time he was symptomatic which was interpreted as "pneumonia," with pO<sub>2</sub> 74. He was misdiagnosed with bacterial endocarditis and treated with IV antibiotics for 8 weeks. He improved during hospitalization but symptoms recurred when he returned home. Subject and wife, both smokers, had abnormally elevated carboxyhemoglobin (COHb) levels out of proportion to smoking; house carbon monoxide levels were undetectable. Wife and children had similar but less severe symptoms, without fevers or sweats. Pet cat's behavior became bizarre.</p>
Duration of Symptoms before Diagnosis	2 years
Microbiological Sampling Results	<p>Bulk sample of bathroom moulding: <i>Aspergillus versicolor</i> (7.0x10<sup>6</sup> CFU/gm), <i>Chaetomium</i> (3.4x10<sup>6</sup> CFU/gm), and <i>Penicillium</i> fungi, with low concentrations of thermophilic bacilli and <i>Thermoactinomyces candidus</i>. Carpet, pad, wall sheetrock and flooring tack strips in water-infiltrated areas of the hallway adjacent to the bathroom: extremely high concentrations (10<sup>6</sup>-10<sup>8</sup> CFU/gm) of <i>Aspergillus</i>, <i>Penicillium</i>, <i>Fusarium</i>, plus spores of <i>Stachybotrys chartarum</i> and thermophilic actinomycetes.</p>
Diagnosis	(?)HP; "SBS-like illness"
Intervention and Outcome	Family was removed from home. All occupants' principal symptoms resolved within 2 weeks.
Comments	Subject screening precipitants (IgG) panel was negative. (N.B. fungal antigens did not match those found in the house). Re-entry challenge test was recommended to establish the diagnosis of HP but landlord prevented further testing from being conducted.
Case	3
Subject(s)	Family: Mother (38), Father (38), 3 children (10, 12, 13)
Description of Home	New, pre-fabricated, modular, 1-story house with basement
Structural Damage to Home	Water heater exploded in basement. Basement wall, ceiling, and carpet were soaked. Family tried to remove water with fans, left wet materials intact.
Clinical Illness	<p>Within one week of flooding, family members developed recurrent "head colds" and "flu." Children developed papular rash over arms and legs and school performance declined. Son with history of mild asthma developed significant worsening of his disease. Mother developed marked behavioral changes, difficulty concentrating and short-term memory impairment, profound fatigue, and persistent facial edema.</p>
Duration of Symptoms before Diagnosis	1 ¼ years

Table I cont.

**Microbiological  
Sampling Results**

Air sampling for culturable fungi:  $>4.6 \times 10^4$  CFU/gm of *Penicillium*, *Aspergillus*, *Cladosporium*, and other fungi. Bulk samples of carpet and pad:  $10^4$ - $10^5$  CFU/gm *Aureobaidium*, *Penicillium*, and other fungi. *Stachybotrys chartarum* spores and conidiophores were identified on virtually all wet sheetrock samples, and in air samples. (Burkard personal impactor).

**Diagnosis**

"SBS-like illness;" asthma exacerbation (son); MCS (mother)

**Intervention and Outcome**

Family was removed from home. All improved except mother who had persistent neurocognitive impairment.

**Comments**

Homeowners' insurance offered only to "patch" water-damaged areas; felt house was habitable. House was foreclosed upon by mortgage holder for failure to occupy premises. Family initiated a lawsuit against their homeowners' insurance company.

**Case**

4

**Subject(s)**

Wife (43) and husband

**Description of Home**

Brand new, double-wide trailer transported from show lot in another state. Modular components were reassembled on-site before occupancy.

**Structural Damage to Home**

Unknown. Author speculates that during transport of two "halves" of house, ceiling insulation (shredded newspaper) and wall insulation (fiberglass) were not properly covered and received water infiltration from rain.

**Clinical Illness**

Within 2 weeks of moving into house, wife developed bizarre personality and mood changes, including becoming verbally abusive to her husband; metallic taste in her mouth; eye irritation; dryness of the nasal mucosa; insomnia; difficulty speaking; fatigue; and difficulty concentrating on simple tasks. Any irritant chemical triggered her symptoms. Both wife and husband developed papular rashes over their bodies. Husband was otherwise asymptomatic.

**Duration of Symptoms  
before Diagnosis**

6 months

**Microbiological  
Sampling Results**

Burkard air and bulk samples yield *Aspergillus*, *Penicillium*, and yeasts in furnace filter, attic insulation and wall insulation after destructive testing 2 years after house was first occupied. House had a yeast-like odor.

**Diagnosis**

"SBS-like illness;" MCS

**Intervention and Outcome**

Family vacated house. Husband improved; many of wife's symptoms resolved but chemical "sensitivity" remained, as well as persistent fatigue.

**Comments**

Homeowners' insurance refused to acknowledge building-related problem, felt house was habitable. House was foreclosed upon by

Table I cont.

	mortgage holder. Family initiated a lawsuit against their homeowners' insurance company to remediate house.
<b>Case</b>	<b>5</b>
<b>Subject(s)</b>	38 year old, single female
<b>Description of Home</b>	One-story, 2-bedroom cottage in high-end residential neighborhood.
<b>Structural Damage to Home</b>	Unknown for 1 ½ years. Discovered "mushrooms" and "black, tar-like substance" above dishwasher in cabinet and on wall. Dishwasher discharge hose had been leaking onto the concrete slab, with water migrating up the wall.
<b>Clinical Illness</b>	Approximately one month after moving in, she developed a variety of unexplained symptoms, including extreme fatigue, axillary pain, maculopapular rash, choking sensation, and hoarseness. Exposure to common chemicals triggered severe symptoms.
<b>Duration of Symptoms before Diagnosis</b>	2 years
<b>Microbiological Sampling Results</b>	High concentrations ( $>4.6 \times 10^4$ ) of <i>Stachybotrys chartarum</i> spores were measured in air (Burkard) samples, while culturable (Andersen) air samples yielded predominantly <i>Aspergillus</i> , <i>Penicillium</i> , <i>Chaetomium</i> , and other fungi. Scotch tape and surface sampling throughout the house demonstrated these fungal species, including <i>Stachybotrys</i> spores on the kitchen ceiling fan blade and in the portable air filter in the living room.
<b>Diagnosis</b>	"SBS-like illness;" MCS
<b>Intervention and Outcome</b>	Subject moved out of house. Most of her symptoms resolved but chemical "sensitivity" remained.
<b>Comments</b>	Homeowners' insurance refused to acknowledge building-related problem, felt house was habitable. House was foreclosed upon by mortgage holder. She initiated a lawsuit against her homeowners' insurance company.

Abbreviations: HP= Hypersensitivity pneumonitis. SBS= Sick building syndrome. MCS= Multiple chemical sensitivity syndrome.

## DISCUSSION

These five cases illustrate important clinical features and issues in the clinical approach to and diagnosis of disorders related to indoor environmental fungal (mold) contamination in the home environment. In all of the cases described, individuals (occupants, or subjects) developed their illness within a few days to weeks of living in a mold-contaminated environment, though in most of the cases the individuals were unaware of the presence or significance of the water damage and/or mold contamination. Moldy odors were not appreciated by all exposed indi-

viduals. All subjects incorrectly attributed their illness to non-environmental causes for many months while continuing to reside in their contaminated houses.

Some individuals developed what was clinically suspected to be a specific BRI, namely HP (Case #1 and 2) or exacerbation of asthma (Case #3). The HP cases were clinical diagnoses which could not be confirmed through antibody testing or tissue biopsy, primarily because of financial and legal constraints. One of two HP cases was clinically confirmed through an actual exposure challenge test in the home environment, with physician-observed evidence of delayed-onset fevers, chills, and dyspnea; bilateral lower-lobe pneumonitis on chest x-ray (superimposed upon a pre-existing, chronic lung disorder that was presumed to be bronchiolitis obliterans); and hypoxemia documented by arterial blood gas. In both of these suspected HP cases, there were other clinical features, such as upper respiratory tract inflammation (sore throat, nasal congestion) and fatigue which were not delayed in onset with exposure to the building environment, and which appeared to overlap with the HP (or HP-like) symptoms.

In contrast to the limited number of BRI's described above, most individuals experienced a "SBS-like" illness defined by irritation of mucous membranes of the eye, nose, and throat; upper and possibly lower respiratory tract irritation, marked by cough, chest tightness, and dyspnea; profound, unexplained fatigue and related constitutional symptoms; evanescent, scattered rashes over the face, neck, torso, and extremities; and/or neurocognitive dysfunction, manifested by difficulty concentrating, short-term memory impairment, mood irritability, and other behavioral disturbances. The distribution and severity of symptoms varied among cases, even within households. Both children and adults were affected.

The diagnosis of an environmental etiology of the fungal contamination became evident in all but one case (#4). Internal and external water damage to each of the home from a variety of causes produced microbiological colonization of building materials in various locations in the occupied space and building envelope. The etiology for illness in Case #4 was never confirmed. It was speculated that the attic insulation (made of shredded newspaper) had not been properly covered during transport and had become wet, such that when the house was sealed, the moist, dirty, cellulose insulation became a source of colonization of fungi. The potential route of exposure to the occupied space was hypothesized to occur through the gap joining the two halves of the "double-wide" house when the (joined) interior space became negatively pressurized as doors in the house were opened and closed.

All of the cases were notable for a significant delay (range: 6 months to 2 or more years) between the onset of symptoms and the time to a definitive diagnosis of environmental disease, including referral to an OEM physician and/or a certified industrial hygienist (CIH). Environmental histories were notably absent from occupants' medical evaluations by their family physicians, internists, and medical specialists. In one case (#2), the (mis)diagnosis of endocarditis (which was suspected because of unexplained fevers and "confirmed" by 1 out of 4 blood cultures positive for what was most likely a skin contaminant during the blood draw) seriously delayed the diagnosis. The subject's (#2) clinical improvement during his hospitalization was deemed to confirm his diagnosis of endocarditis, when in fact he improved clinically because he had been removed from his home environment. Failure to obtain an adequate, or for that matter any occupational and environmental history continues to be problematic for physicians in primary care and medical specialties (McCurdy et al. 1998). In buildings with rel-

atively few occupants, such as these residential cases, illness among a small number of occupants may not raise occupants' suspicion of an environmental etiology. The delay in recognition of an environmental etiology by the occupants themselves may have been due in part to the fact that they spent so much time in their homes that they did not notice an environmental relationship to their symptoms. Individuals who present to physicians with a symptoms-complex of persistent or recurrent rhinitis, sore throat, cough, chest tightness, and fatigue are often (mis)diagnosed as having (viral or bacterial) upper respiratory tract infections, asthma, or "allergies," and are often (inappropriately) treated with oral antibiotics. Alternative (differential) diagnoses or etiologies, even after multiple visits for the same complaints, are typically not considered. Neurocognitive symptoms, as described above, differentiate the mold-related environmental syndrome from these common infectious respiratory tract ailments, but are often not elicited by physicians or volunteered by patients in the presence of the other prominent, "organic" symptoms which brings these patients to seek medical attention.

Most occupants in this residential case series developed symptoms consistent with those described in cases of "sick building syndrome," namely a constellation of mucous membrane (eye, nose, throat) irritation; upper and possibly lower respiratory tract inflammation or irritation (rhinitis, nasal congestion, hoarseness, cough, dyspnea, chest pain); prominent fatigue, headache, and malaise (constitutional); sometimes skin lesions (erythematous, papular rash); and neurocognitive dysfunction (difficulty concentrating, behavioral changes, fatigue, headache), which occur upon or shortly after entering a particular building, and which improve after leaving the building. Physical examination and laboratory test results were unrevealing late in the course of nearly all cases (except #1), particularly with regard to objective evidence of lower respiratory tract disease. Rashes, nasal mucosal inflammation, and wheezing in (pre-existing) asthmatics were the most consistently documented examination findings.

Currently available immunological tests such as fungal IgE and IgG antibodies, have been shown to be of low sensitivity and specificity as markers of exposure or disease in non-infectious, mold-related illness in the absence of a specific (i.e., Type I, III-, or IV- immunologically-mediated) BRI such as asthma or HP (Hodgson et al. 1998; Johanning et al. 1996; Malkin et al. 1998; Rylander 1994). Even when the clinical presentation is consistent with HP, the diagnosis of HP is not always able to be confirmed immunologically, particularly when more than one potentially immunogenic fungal (or bacterial) antigen is detected environmentally. The finding of unexplained, elevated carboxyhemoglobin levels in one case (#2) warrants further exploration of its clinical significance and potential as a marker for fungal bioaerosol exposure.

The role of certain fungal genera and species, particularly toxigenic fungi which thrive indoors in areas of high water activity such as on water-saturated gypsum board and ceiling tiles, is becoming increasingly implicated as the cause of certain building-related illness that is clinically indistinguishable from sick building syndrome (Hodgson et al. 1998; Johanning 1996; Sudakin 1998). Current theories about the pathogenesis of this mold-related illness suggest that symptoms may be caused by a combination of an allergic-type response to fungal components, and/or systemic toxicity or neurotoxicity due to mycotoxins and/or microbiological volatile organic chemicals (MVOCs) through several potential mechanisms (Sorenson et al. 1987). The clinical illness does not appear to represent an infectious pulmonary or systemic fungal disease, nor does it appear to be mediated through a classic, immediate (IgE-mediated)



allergic mechanism. Symptoms are not always consistent among similarly exposed individuals, and some exposed individuals' illness often become more severe over time, thus impeding an estimation of whether a exposure-response relationship exists. Furthermore, occupants' subjective complaints often are not confirmed by objective evidence of organ dysfunction, particularly in the case of possible obstructive lower respiratory tract disease (Muzi et al. 1998). Limitations in sampling, quantification, and speciation of indoor fungal contaminants also confound exposure assessment and estimation of dose-response and immune-response relationships.

These limitations in the understanding the pathophysiology of disease, and inconsistent approaches to case definitions in epidemiological investigations, have impeded the general acceptance and definition of a non-infectious, mold-related disease entity within the medical and scientific communities (Smoragiewicz et al. 1993). The author therefore proposes the term "NIFIES" (pronounced "knife-eez," for Non-Infectious Fungal Indoor Exposure Syndrome) to designate and recognize this specific BRI caused by indoor mold exposure in residential and commercial buildings. As clinical, epidemiological, microbiological, and toxicological research elucidates a specific disease mechanism, this syndrome may be replaced by a more specific disease appellation, and may replace the term "SBS" as well.

Three of five cases in this report had *Stachybotrys chartarum* identified as an indoor contaminant. The presence of *Stachybotrys chartarum*, which has received considerable attention from one case associated with dramatic health effects unrelated to SBS-like symptoms (Montana et al. 1997), has been observed in numerous occupational and residential cases where one or more species of toxigenic fungi have been environmentally detected in high concentrations on building material surfaces and/or in air (Sorenson et al. 1987). The clinical significance and human toxicology and immunology of *Stachybotrys chartarum* and other fungal trichothecene mycotoxins remains incompletely understood at present (Fung et al. 1998). The clinical illnesses in these cases where *Stachybotrys* was identified can therefore not be solely attributed to this particular organism in the presence of other genera of toxigenic fungi such as *Penicillium*, *Aspergillus*, and *Chaetomium*.

Most of the occupants who were diagnosed with a BRI or "SBS-like illness" (or, as suggested above, NIFIES) had marked clinical improvement after removal from the residential environment. Remediation of the water-damaged and mold-contaminated house was attempted in only one (#1) case, with paradoxically greater indoor contamination created by lack of appropriate containment during remediation of mold-contaminated building materials. However, even after occupant removal, in three cases (#3,4,5) some occupants continued to experience primarily neurocognitive symptoms, though most of their other respiratory, skin, and mucous membrane symptoms had subsided. Two of these individuals, both female, experienced recurrent, predictable episodes of triggering of neurocognitive and other symptoms upon exposure to a variety of environmental chemicals and substances. This clinical scenario is consistent with the clinical entity of multiple chemical sensitivity (MCS). Like SBS, MCS remains a controversial disorder whose etiology remains poorly understood (Simon et al. 1993). This clinical observation nevertheless suggests that MCS may be a final common pathway for environmental insults that are not classically due to "toxic" chemical exposures. This finding of "bioaerosol-induced" MCS, albeit unexplained, may represent another possible long-term complication

from exposure to airborne concentrations of certain indoor fungi. Many of the scientific problems and issues surrounding MCS research and clinical validation are applicable to these cases, since they can only be studied retrospectively and lack any baseline neurobehavioral data for comparison (Fiedler et al. 1997).

Finally, from a practical and clinical standpoint, all of the cases were notable for conflicts caused by homeowners' or other insurers delay in or refusal to acknowledge the health hazards of indoor fungal contamination, the authorization of timely and qualified medical evaluation of occupants, and the provision of insurance coverage and relief for these homeowners or occupants. Legal intervention ensued in all of the cases. Two cases (#2 and 5) involved occupants who were not aware of water damage which had pre-existed their occupancy of the home. In one case (#2) which involved controversy over the issue of whether the medical condition was work-related, the workers' compensation process required lengthy legal appeals to substantiate a complete OEM investigation by the author, further delaying the investigation of the subject's residential environment and ultimately the correct diagnosis for many months. Once the environmental etiology in Case #2 was finally established, the property owner evicted the tenant and then demolished the contaminated portion of the house (evidence) before a diagnosis of HP could be confirmed. Reasons proffered by homeowners' insurance representatives (including lawyers and retained medical "experts") included refusal to recognize non-infectious, mold-related illness as a "valid" medical condition, and lack of "objective evidence" such as toxicological tests, allergy tests, or lung function tests to "prove" that the exposure occurred and that it caused the clinical illness. Insistence by insurers for industrial hygienists to "test for chemicals" (e.g., Case #4) when no such chemical exposures are evident from history and building inspection also delayed appropriate microbiological investigation. Finally, lack of familiarity with the role of a qualified OEM physician in conducting a building investigation appeared to be common factors in some insurers' decision to delay or deny a clinical investigation. These problems led to significant emotional and financial strain on affected occupants, particularly those who were forced to abandon their homes and continue paying mortgages.

## CONCLUSION

Exposure to fungal contamination of water damaged houses and other buildings causes an SBS-like symptom complex and, to much lesser extent, specific, recognizable building-related diseases. Diagnosis and treatment of mold-related indoor illness is often delayed due to problems with medical evaluation, insurance claim acceptance, and delays in obtaining qualified, focused investigation of the home environment. Removal from (or remediation of) the contaminated environment usually but not always results in clinical improvement of affected individuals. The term "non-infectious, fungal indoor exposure syndrome" (NIFIES) is proposed to define the illness described herein, as well as in other similar cases reported in mold-contaminated occupational and environmental settings, as a unique disease entity. Further clinical, epidemiological, microbiological, and other scientific research is needed to elucidate the mechanisms of disease, and to identify and quantify sources and routes of human exposure to fun-

gal bioaerosols. The determination of whether NIFIES is indeed the same clinical entity as sick building syndrome also awaits these advances.

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